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THE INVESTIGATION OF "PHYSIOLOGICAL" PLANT DISEASES[1]

RALPH E. SMITH

In the development of plant pathology there has always been a tendency to maintain a certain, semi-specific class of diseases as a sort of catch-all for those of unknown and obscure cause. Insects, being the most obvious, were first most thoroughly studied; when fungous parasitism became known, but before its scope was thoroughly comprehended, all troubles and diseases wherein no insect could be found were likely to be ascribed to fungi, "fungous disease" meaning any obscure trouble. As parasitic fungi became better known and methods for their detection and study developed, the term fungous disease became a well defined one. Then came a tendency to ascribe the mysterious troubles of unknown cause to bacteria and "bacterial disease" came often to mean anything not caused by insects or fungi.

Again science has progressed; methods of detecting and studying bacteria pathogenic to plants have been standardized and there is now no reason to ascribe to bacteria any but diseases which can be positively proven to be due to such organisms.

In the meantime there has gradually accumulated a list of diseases, many of them very characteristic ones, for which no specific cause has been found and which do not appear to have any mode of parasitic etiology with which we are familiar. For these the term "physiological" or "non-parasitic" disease has come into use, the idea being, in a general sense, that the trouble is due to an autogenous functional disturbance rather than to the presence of a foreign organism. Included in this class there is at present a variety of peculiar troubles, in some of which there will undoubtedly yet be proven a connection with parasites of recognized

[1] President's address at the meeting of the Western American Phytopathological Society, Corvallis, Oregon, Dec. 30, 1914.

groups. In fact, some of these diseases are continually being taken from the doubtful class and proven to originate in some form of parasitism. A conspicuous example of this is the gummosis of the lemon, a disease which was described by the writer and O. Butler (1908) as a typical, physiological one. A few years later, Fawcett (1913), working at the writer's suggestion and in his laboratory, proved conclusively that the effects produced by this disease are not of autogenous origin but depend absolutely for their inception upon the presence in the tissues of a parasitic fungus. Other physiological diseases may be simply injuries or functional disorders due directly to unfavorable or injurious conditions, although the more specific of the diseases commonly thought of in this connection seem to be more complicated than this.

It is not entirely easy to characterize or define with accuracy the present conception of a physiological disease, although probably it is fairly uniform in the minds of most plant pathologists. Some conspicuous examples of these diseases are peach yellows, bitter pit or baldwin spot of the apple, blossom end-rot of the tomato, exanthema and several other diseases of citrus trees, curly top of the sugar beet, leaf roll, internal brown spot and other diseases of the potato, little leaf of many kinds of trees in California, various types of chlorosis and gummosis, and apple rosette of the western United States. The list might be considerably extended to a point where it would be impossible to draw the line between "physiological" diseases and direct, unquestioned injury from unfavorable or injurious soil or climatic conditions. No argument is needed to show that these diseases have no proven relation to each other. A definition of a class of disease to include them all would amount to something like this: A physiological plant disease is one with specific, well marked characteristics in which no causal relation has been established with any pathogenic organism and with symptoms which do not appear like the effects of parasites of any of the usual kinds. This amounts to saying that it is a disease in which the cause is not known but which does not look like a parasitic one. There is, however, the further conception in these cases to the effect that the trouble is due to a functional or metabolic disturbance in the plant, more or less autogenously incited or maintained. The definition is and must be largely a negative one, since, as we shall soon show, no disease is known positively to be of any such nature.

Good examples of the theory of physiological disease in plants are afforded by some of the various conceptions which have been developed concerning the mosaic disease of the tobacco. Hunger (1905) says: "I consider the virus of the mosaic disease to be a toxin, which is continually being secreted in the tobacco plant in connection with the metab-

olism of the cells and which, under normal conditions, exerts no effect; during too active metabolism it accumulates and then causes such disturbances as the mosaic leaf mottling. I assume that the toxin of the mosaic disease, which is primarily produced by exterior irritation, is able by penetrating into the cell to exert a physiological irritation with the result that the same toxin is secondarily formed there again; in other words, the mosaic disease toxin possesses the property of acting in a physiological, autocatalytic manner." Woods (1899), Heintzel (1899), and others demonstrated that the relative activity of various enzymes in the diseased leaves is abnormal, the former (1900, 1902) formulating the definite theory that the disease is due to an inhibitory action upon starch hydrolysis and translocation, induced by an excess of oxidizing enzymes. Chapman (1913) says: "The direct cause may be found in the presence of free hydrogen peroxide or some other residuary by-product of plant metabolism which has been allowed to remain unassimilated in the cells of the leaves through excessive development and retardation of certain enzymes." He views with particular suspicion the deficiency of catalase which he finds in diseased leaves, seeing in this a possible lack of hydrogen peroxide reduction and consequent injurious accumulation of the latter.

The fact that the tobacco disease is known to be infectious through contact of the juice of diseased plants with healthy plants has not discouraged these investigators in their idea of a non-parasitic origin. They assume an infection by "causative enzymes," similar to that by bacteria or other living organisms, or ignore this phase of the matter.

The present writer (1902) showed in the aster yellow disease a lack of diastatic and oxidizing activities in diseased leaves, together with an excess or accumulation of starch and metabolic by-products such as tannin and organic acids. Sorauer (1909) sees in typical physiological disease "A disturbance of function, or, in other words, a different direction in the molecular motion to which we must ascribe all metabolic processes." In gummosis Beijerinck and Rant (1906) held that "The cells of the cambium contain a cytase, which, while they remain alive, is unable to attack the cell wall owing to the semi-permeability of the protoplasm. When, however, any cells of the embryonic wood are killed by penetrating hyphae, traumatisms or a toxic agent such as mercuric bichloride, the contained cytase diffuses out and is able to attack the walls of the circumnambient, healthy cells, which become gummose and finally dissolve away. More cytase is thus released, attacks other healthy cell walls, and in this manner the gum pocket is formed" (translated by and quoted from Butler (1911)). Others have held similar views. Butler (1911) states that "The autogenous form of gummosis appears to be

confined very largely to the cherry and the lemon and is induced when vigorous growth is accompanied by an excess of water in the substratum." "Gummosis degeneration is to be explained in some other way than by assuming the pathological action of an enzyme. . . . . I am inclined to believe that were the genesis and development of the cell wall better known gummosis could be explained on a purely physico-chemical basis." Other physiological diseases have been ascribed directly to excess, lack or improper proportion of certain chemical elements in the soil, climatic effects, varietal "running out," inherited weaknesses caused by previous generations growing under unfavorable conditions, biological disturbances in the soil, disturbed osmotic pressure in the plant tissues, and all manner of environmental and functional irregularities.

The writer is attempting a systematic study of some of the most important, supposedly physiological diseases with the purpose of learning the true nature of these troubles and of adding any possible information to our fundamental knowledge of the etiology of plant disease. In carrying out this study and in considering the choice of the most profitable lines of procedure, we have been much impressed with certain fundamental considerations suggested by previous work on these diseases, which may be discussed at this point.

In the present explanations of all the prominent physiological diseases, it is a fact without exception that there is wanting a primary, inciting influence to inaugurate and maintain the structural, functional or enzymatic disturbances which are so easily demonstrated. As to the etiology of even the best studied of these diseases it may be said without exception that in no case has such inciting influence been discovered. The explanations advanced have been theoretical from beginning to end and no proof whatever has been even attempted that the chemical, enzymatic and functional irregularities found represent cause rather than effect or that these conditions are limited to the specific disease under consideration.

The assumption of infection by enzymes or through the transmission of molecular motion has absolutely no foundation of knowledge, is totally at variance with established principles and scarcely merits discussion; it seems more reasonable to seek in such cases the presence of an obscure parasite, as is constantly being done with good results in animal pathology.

We have no absolute proof of the nature of any of the diseases listed above, and no one has ever succeeded in inciting any of them with a specific factor by any method which excluded all possible influence except the one to which he attributed the disease. Indeed we may fairly question whether any specific plant disease has ever been reproduced by any single, well segregated agent except a parasite. We do not assert that real, non-parasitic plant diseases may not or do not exist, but simply

that none have been proven and none of those which are commonly thus classified have been thoroughly studied in some of their most important features. Metabolic diseases are accepted by our more advanced brethren of human pathology, which shows that such functional disorders are not impossible.

Parasitism. Our knowledge of parasitism, either in plants or animals, is by no means exhausted. It is scarcely twenty years since Erwin Smith challenged the skepticism of the European world in regard to the existence of bacterial plant diseases. Still more recent has been most of the development of knowledge of pathogenic protozoa, spirochaetes, sporozoa and filtrable viruses in animal pathology. With these examples before us, who shall say that organized, living parasites of wholly unknown types do not exist? Indeed we do not need to resort to such ultra-considerations as these to show the possibilities of overlooking a parasite, inasmuch as every pathologist realizes the difficulties which often exist in such work.

In the present status of plant pathology the following broad principle cannot be denied: "*There is no single instance known of a positively demonstrated inciting cause of any specific plant disease except a parasite.*"[2] We have theories, indications, appearances, and effects which point otherwise, but of proof we have none in any case. Therefore, in the investigation of diseases of obscure etiology, should we not first of all proceed from the known to the unknown, disregarding general appearances, indications and theories based on previous, more or less limited experience, and exhaust to the utmost the possibilities of parasitism before confidently declaring our disease to be non-parasitic? The writer speaks with more assurance on this point from having been himself one of the worst offenders. In regard to the so-called yellows of the aster, he stated with assurance (1902), but with no more confirmatory information than is given in this paragraph:

"It is natural in a plant disease to look for some parasitic organism as the cause. In many respects the appearance and nature of this disease lead one to suspect some such origin, but it may be said at once that the most careful search in all parts of the plant has failed to reveal anything of the sort. That the trouble is purely of a physiological nature, due to some perversion of the normal functions of the plant, can scarcely be doubted." At the present time we would be loathe to discuss the extent of the "most careful search" which proved so conclusively that no parasite is present in this disease!

[2] Specific means here, of course, something more complicated or obscure than direct traumatic effects.

Again in discussing gummosis of citrus trees we stated (1908): "It may be said here without extended discussion that in no case have we been able to recognize or demonstrate the presence of any fungus, bacterium or other parasitic organism as the cause of any form of citrus gum disease. . . . . We feel safe in concluding that the diseases hereafter described are of a physiological or autogenous nature. . . . . The appearance and occurrence of the diseases is not such as to point to parasitic infection as the primary cause." And yet a few years later Fawcett (1913), in the same laboratory, proved the most prevalent type of the disease to be due to an easily isolated fungus of a genus and species first described by the writer himself. In regard to the curly top of the sugar beet, we stated (1907): "The conclusion reached and substantiated by a great variety of field and laboratory investigations, observations and experiments extending over two seasons, has been that the trouble is not brought about by any parasite or organism, but is rather due to a derangement in the normal functions of the plant. . . . . The disease is one of a number of so-called physiological diseases." We hope soon to be able to offer proof that this disease is strictly of parasitic origin, from investigations now going on.

Having before him these examples of his own errors in this respect, the writer is inclined to be over-cautious perhaps in accepting similar statements from others. Are we certain that the possibilities of parasitism have been exhausted or even thoroughly considered in the tobacco disease,[3] bitter pit of the apple, blossom end-rot of the tomato, peach yellows, aster yellows, and other diseases of this class? We say this with no criticism or disparagement of the work done upon these diseases except as to its completeness upon this one point.

Difficulty in proving parasitism. The ordinary methods of demonstrating parasites are well known, consisting of direct examination with the microscope with or without staining, making of cultures from diseased tissues, and attempts to produce infection. The difficulties of proving parasitism in individual cases are many and varied. There is, first of all, theoretically at least, the possibility that types of parasites may exist of which we have no knowledge or conception. If, however, we add the more recently discovered types of animal pathogens to our usual list, we may busy ourselves for some time before exhausting present resources.

A second source of difficulty or error lies in the fact that the parasite may be located in another part or organ than that in which the visible

[3] Allard's recent work (1914) shows almost certainly that the inciting factor in this disease is of the nature of a filtrable virus, as suggested by several previous investigators.

or most striking effects of the disease appear. In the "mottled leaf" disease of citrus trees Thomas (1913) has shown the existence of a very abundant parasitic nematode of an entirely new species upon the roots of affected trees, where previous investigators had not made a careful examination. Humphrey (1914) has just shown proof that the "western blight" of the tomato, previously considered a physiological disease, is due to a fungus attacking the finest rootlets under certain conditions. Many similar instances suggest themselves. Therefore all parts of the plant must be thoroughly studied and searched for a possible parasite and not simply the part where the most prominent effect is seen. Criticism on this point is justified in almost all the work that has been done on physiological diseases.

The parasite may attack the plant only at a certain or for a limited period and disappear before the symptoms of the disease are outgrown or even before they are well developed. This applies more especially to insects, worms, and such animal parasites than to fungi or bacteria.

Parasites are very often refractory to the usual staining, culture or other methods of detection, and there may be some which are impossible to detect with certainty in the host tissues and many we know will not grow in a culture by any of our present methods. The most exhaustive researches by the latest methods of bacteriology, parasitology and cytology should be made before the search for visible parasites or foreign bodies of any kind in the tissues is concluded. The mere fact, too, that cultures have been attempted on the ordinary media without success is no proof that parasitism does not exist. In reporting results the investigator should state clearly the media and methods which he has used unsuccessfully and avoid such statements as "various media" or "the usual media" were employed, as though this settled the matter.

Infection takes place in some cases under such limited circumstances that one may see or culture the right organism but not hit upon the necessary mode or time of infection. The loose smuts and some of the insect-transmitted animal parasites are examples of this. Also curl and California blight of the peach.

Again, difficulty in proving parasitic etiology may easily occur in the case of diseases where certain specific lowering of or change in the resistance of the host, or an invigoration of the parasite is necessary before infection can take place. Certain degrees of temperature, soil or atmospheric humidity, light intensity, nutritional relations, and other factors may accomplish this.

Lastly, as regards parasitism, the mental attitude of the investigator, a preconceived notion that certain diseases are non-parasitic, has probably

done more than anything else to discourage thorough work along this line.

Let us repeat that we would by no means be understood as holding that all plant diseases are necessarily parasitic ones. We particularly deprecate the burdening of the literature of some of these diseases with poorly established, easily disproven claims of supposed parasites, as has so frequently been done. We suggest only that none of the so-called non-parasitic troubles has been sufficiently investigated in this respect, that from time to time a careful investigator shows that his predecessors have overlooked something, that there is at present no established group of non-parasitic diseases for the kind of troubles which we are considering, that we should not abandon too soon the known for the unknown, and that the investigator should at least, in publishing his results, state exactly what methods he employed and the attendant circumstances, avoiding general statements to the effect that the search for parasites has been exhausted.

Symptomatology, histology, cytology, diagnosis. In studying a new or poorly known disease there is nothing more necessary than that we should have definite means of detecting the disease in all stages and sharply distinguishing it from all other effects. This can always be done where a specific organism can be detected and to a certain extent is possible by means of gross, morphological changes and symptoms. Such symptoms, however, are rarely adequate and may not appear at all in the earliest stages of the disease, when in many cases it can be studied to the best advantage. Color changes, irregularities of growth or form, premature or partial death and similar symptoms are all of value and should be carefully looked for and described, especially when found with certainty to be characteristic of the specific disease under consideration. But preceding and causing such symptoms there must always be present functional or tissue disturbances which may often be capable of demonstration by histological means. Such symptoms, moreover, are of great value in studying the etiology and development of obscure diseases. Animal pathologists long ago arrived at *cellular pathology* as the last resort in diagnosis, and the plant pathologist can do no less if he would solve his most obscure problems. Through all our discussions of "enzymatic disturbances," "molecular motion" and "functional disorders," and our empirical applications of chemicals, fertilizers, water and various other substances in a blind effort to stumble onto something of promise, the fact remains that in no single instance (except to a certain extent in gummosis) do we have any accurate or comprehensive idea in these diseases of the cytology or histology of affected plants. In some cases we know that certain tissues die or are discolored but that is about all.

Let one read the recent edition of Mallory's Pathological Histology, written from the standpoint of human pathology, and he will realize the value of such knowledge and its deficiency in plant pathology. Let him study Erwin Smith's 1912 photomicrographs of crown gall and ponder on our knowledge of peach yellows, judged by the same standard. In the study of obscure diseases we should get at the very beginning a comprehensive, accurate knowledge of all pathological changes of structure in the whole plant so far as they can be detected by histological or any other means. We should attempt to detect the very first inception of the disease and follow its effects throughout. Knowledge of general effects upon tissues is not sufficient but we must go down to a clear conception of the condition of the cells and their contents. This necessitates a similar study of the normal plant for purposes of comparison and may often involve considerable difficulty in obtaining specimens known to be absolutely free from the slightest degree of the disease. Not only in the case of obscure and physiological diseases is such study needed, but likewise with plants affected with diseases or lesions of known cause, we need a vast amount of accurate work upon the specific structural and functional effects of all pathogenic influences; each bit of information of this sort added to our store of knowledge furnishes one more possible clue to the detection of unknown causes of disease by comparative study of effects.

One of the most important phases of the kind of work just discussed has been thus far almost entirely neglected in the investigation of physiological diseases. *Histological study should not be confined to the part of the plant where gross lesions or abnormalities appear, but should include every part and organ.* Our meagre knowledge of the pathological histology of bitter pit and of blossom end rot, for instance, is confined to the fruit, where prominent, gross symptoms appear. Nothing is more desirable than a careful, histological study of the whole plant in these cases. Mr. A. Boncquet, a graduate student in our laboratory, has found that in the case of the apple disease lesions occur in the vascular tissue at least as far back as the stem, and in fruit not yet fully grown. Comprehensive work of this sort will undoubtedly eliminate some theories and strengthen others as to the etiology of these diseases. Similarly with parasitic diseases, traumatisms or intoxications, we need to know the effects, if they exist, upon the whole plant and not simply those in the vicinity of the initial disturbance or the most obvious effects. Such accurate, complete knowledge would undoubtedly aid greatly in the elucidation of our obscure problems.

The investigator should search especially for *characteristic* symptoms, for *initial* ones and for the *totality* of significant effects, so that he can

proceed to describe the symptomatology and pathogenesis of the disease in such a manner that future investigators and students may have the minimum of uncertainty in identifying the same disease.

Pathological physiology and biochemistry. The biochemical work already done upon some of our obscure diseases, as well as upon some of known parasitic origin, has revealed great possibilities for comprehensive, thorough research in this direction. Technical knowledge of the methods and status of similar work in animal pathology is needed for such work. Wells' Chemical Pathology and Zinsser's Infection and Resistance are of great value to the investigator in this connection, as well as being most readable to any person interested in pathology.

A danger of fragmentary biochemical work in connection with these diseases lies in the lack of discrimination between cause and effect of the disease, which characterizes most of the work which has been done thus far.

Conclusion. In conclusion the writer may summarize his ideas on this subject as follows:

There is no such thing as an established group of physiological or non-parasitic plant diseases for the kind of troubles which we have been considering. There is among them not even a single, well proven example. They are all more or less obscure diseases of unknown etiology, which, for one reason or another, have not yet been accounted for.

In all diseases found to be really infectious, either through inoculation with plant parts or juice, by budding or grafting or by any transmission method producing true infection in normal plants, it is best to assume a parasitic factor.

The only positively known inciting factors in plant disease (excluding direct traumatisms) are parasites. In the investigation of diseases wherein an obscure, ever active, inciting factor appears to be present, we should therefore proceed from the known to the unknown, endeavoring within reasonable limits to exhaust every known phase of parasitism before assuming conceptions of no established importance.

Nothing is more important than a thorough knowledge of the pathological histology and cytology in these diseases, although such knowledge is at present extremely lacking.

Biochemical work is likewise of great importance but may easily be so superficial as to result in false and misleading conclusions.

The study of these diseases should include every part of the plant and not simply those where gross symptoms or lesions appear.

The most substantial proof should be obtained before final conclusions are drawn. Tentative or incomplete results may be worth publishing but should be clearly stated as such, with the attendant methods

and circumstances, giving the reader an opportunity of drawing conclusions of his own. Many facts but few conclusions is a safe rule in such cases.

A thorough knowledge of pathological histology and chemistry of diseases of known cause is equally important, especially for the elucidation of obscure troubles by comparison.

The greatest progress in fundamental knowledge of plant disease can be made only by pathologists; not mycologists, physiologists, bacteriologists, cytologists, or chemists, but men who combine an intelligent interest in and appreciation of these elements in their relation to the study of disease.

UNIVERSITY OF CALIFORNIA
BERKELEY, CALIF.

BIBLIOGRAPHY

WOODS, A. F. 1899. The destruction of chlorophyll by oxidizing enzyms. In Centbl. Bakt. u. Par., abt. 2, **5**: 745–754.

1900. The inhibitory action of oxidases upon diastase. In Science, n. ser. **11**: 17–19.

1902. Observations on the mosaic disease of tobacco. U. S. Dept. Agr., Bur. Pl. Ind., Bul. 18: 1–24.

HEINTZEL, K. 1899. Contagiöse Pflanzenkrankheiten ohne Mikroben etc. Inaug. Diss. Erlangen.

SMITH, RALPH E. 1902. Growing China asters. Hatch Exp. Sta., Massachusetts Agr. Coll. Bul. 79: 1–26.

1907. Beet blight investigation. In California Agr. Exp. Sta., Bul. 84: 240–241.

—— AND BUTLER, O. 1908. Gum disease of citrus trees in California. California Agr. Exp. Sta., Bul. 200: 235–270.

HUNGER, F. W. T. 1905. Untersuchungen und Betrachtungen über die Mosaikkrankheit der Tabakspflanzen. In Zeitschr. Pflanzenkr. **15**: 257–311.

BEIJERINCK, M. W. AND RANT, A. 1906. Sur l'excitation—gommeux, etc. In Arch. Neerland. Sci. Ex. et Nat., ser. 2, **11**: 184–198.

SORAUER, P. 1909. Handbuch der Pflanzenkrankheiten. (3rd ed.), 1: 678.

BUTLER, O. 1911. A study on gummosis, etc. In Annals of Botany, **25**: 107-153.

SMITH, ERWIN F. 1912. The structure and development of crown gall. U. S. Dept. Agr., Bur. Pl. Ind., Bul. 255: 1–60, pls 1–59.

THOMAS, E. E. 1913. A preliminary report of a nematode observed on citrus roots, etc. California Agr. Exp. Sta. Circ. 85: 1–14.

FAWCETT, H. S. 1913. Two fungi as causal agents in gummosis of lemon trees in California. In California State Com. Hort. Monthly Bul. **2**: 601–617.

CHAPMAN, G. H. 1913. "Mosaic" and allied diseases, etc. In Massachusetts Agr. Exp. Sta. Ann. Rept. **25**: 41–51.

ALLARD, H. A. 1914. The mosaic disease of tobacco. U. S. Dept. Agr. Bul. 40: 1–33.

HUMPHREY, H. B. 1914. Studies on the relation of certain species of Fusarium to the tomato blight of the Pacific Northwest. Washington Agr. Exp. Sta. Bul. 115: 1–22.

LIGHTNING INJURY TO POTATO AND COTTON PLANTS

L. R. Jones and W. W. Gilbert

With Plates VIII and IX

The fact that lightning often causes serious injury to trees of various kinds is well known, and botanical literature contains many articles describing the nature and extent of such damage in all parts of the world. References to injury of field crops or other herbaceous plants by lightning are much less frequent. Indeed we find no published records of this nature from America and few from Europe. We believe, however, that such injuries are not uncommon, at least on the two crops named in the title. During the last two years the authors have been observing these independently, the one (Jones) on potatoes in Wisconsin, the other (Gilbert) on cotton in South Carolina. The likeness of the phenomena makes it seem wise to combine discussions.

LIGHTNING-STRUCK COTTON

It is a matter of common knowledge to cotton growers of the southern states that cotton fields are liable to injury by lightning. Two cases of such injury came to the attention of the junior author in adjoining fields at Monetta, South Carolina, in the summer of 1913. In both cases the injury was noticed shortly after a severe electrical storm which occurred at midday on August 3, and it was noted by the owners that the lightning struck in the locality where the injury was later discovered. The fields were visited and the plants examined by the author, August 28. At the time when struck by lightning the plants were nearly full grown, two to four feet high, the earliest bolls practically mature, but none opened as yet. The soil was light sand. In the first case examined, all the plants were killed over an area some three rods in diameter. We were told that following the storm the leaves on these plants promptly wilted, died and blackened but remained clinging to the plants. The bolls soon began to turn pale, yellow and dry up, but failed to open. By observations in other years and conversation with experienced cotton growers, we have learned that a dead spot in the field as described above represents the common type of injury both as to size and symptom. Such lightning-struck spots in a field are easily distinguished even at a distance by their round shape and the almost coal-black color of the dead leaves. If the lightning

strikes after cultivation ceases, as in the above case, this area soon grows up to cocklebur or other weeds (pl. VIII, fig. 1). Between the central area, where the plants were all dead, and the surrounding uninjured field, there was a zone of plants showing diminishing degrees of injury. Some of these plants lived for days or weeks. The leaves meanwhile slowly changed their color to a yellow or brick-red, suggestive of the autumnal tints of certain trees, drooped and finally blackened and died. Figure 2, plate VIII, shows such a plant with leaves and boll still clinging to the branches.

On pulling up some of the dead plants it was noted that in all cases the immediate effect of the injury was most pronounced on the stem and root system from the surface of the soil downward. The bark here was dried, sunken and darkened, and, at the juncture of this with the uninjured bark above, there was a slight shoulder or ridge as shown in plate VIII, figure 3. This bark injury extended entirely around the stem. Where the injury was less severe, so that the plant continued longer alive, a distinct callus was formed beneath the bark just above the dead tissue. This is well shown in figure 4, plate VIII, where the pronounced callus growth, entirely surrounding the stem, has ruptured the bark on the dead portion at several points forming cracks of one-fourth to one-half inch in width. In this instance, also, a new sprout pushed out above the callus to a length of some two inches. The moist soil conditions following the storm prolonged the life of the plant for some time in spite of the injuries to the root system, but as soon as the surface soil again dried out the plant died. In a few cases, the inside of the stem of these lightning struck cotton plants was discolored in a manner resembling that characteristic of the wilt or Fusarium disease, but this malady does not occur within several miles of this place. Moreover, examination of these stems showed no Fusarium. No cracking of the bark or splitting of the wood was observed as a direct effect of the lightning injury, nor was there any evident disturbance of the soil about the plants.

Two other cases on an adjoining farm differed from the one already described in that not all of the plants within the central portion of the lightning-struck area were killed. Instead, the injury occurred in smaller groups, two to six plants in a place; some dying immediately and turning to the characteristic black color, others changing more slowly, the leaves reddening first, while still other adjacent plants showed no injury, whatever. The injured plants showed symptoms identical with these from the typical lightning-struck area on the adjoining farm. There seems no doubt that their death was due to lightning, although no fully satisfactory explanation is offered for the difference in phenomena. The close proximity of a small persimmon tree in one of these latter fields and of a hedge in the other may have had some influence on the strength and distribution of the electric current.

LIGHTNING-STRUCK POTATOES

During each of the last three mid-summers, 1912–1914, the senior author has been receiving reports of the sudden death of the plants in sharply defined, somewhat circular areas or spots, in potato fields. In every case reported these have appeared in July when the Wisconsin potato is at its highest vegetative vigor. Moreover, this is the season when thunder storms are most prevalent here and in numerous cases the farmer attributed the loss to lightning. Nevertheless, when the first cases were reported to us, we made critical examinations of the afflicted plants, both in laboratory and later in the field until satisfied that there was no parasitic organism associated with the trouble. Experience teaches the pathologist to be cautious in accepting popular opinion as to the causal relation of lightning or electrical currents with mysterious plant maladies. We have, therefore, been gratified when during each of the last two summers convincing evidence has come to us that lightning is the cause of this trouble. We will cite one case in detail.

John Cherf, an experienced and successful potato grower at Antigo, Wisconsin, had in 1913 a fine field of uniformly healthy, vigorous potatoes growing immediately adjacent to his residence. On or about July 12 while watching a severe electrical storm from his window overlooking this potato field, there came a blinding flash of lightning immediately before him. As soon as the storm passed he made examination in the direction of the flash and found evidence that the bolt had struck in his potato field about twelve rods from the house. The potato tops appeared broken and dishevelled over a small area, and upon drying off within 24 hours, they wilted and died. When examined by the senior author some three weeks later, the plants were all dead and dried up over a circular area about twelve feet in diameter. Immediately bordering this dead area, many of the plants showed injury, but aside from this the plants over the entire field showed a splendid stand of vigorous growth. The injured plants, still living, showed partial collapse and death of the stem from a little below the ground line upward through one-half their length, more or less. The tips were living and below ground the base of the stem, roots, stolons and young tubers seemed uninjured (cf. pl. IX, fig. 1). Indeed in some cases new sprouts were starting up from the bases of stems of which the aerial parts were nearly or quite dead. In several other fields examined the conditions were essentially the same except for variation in diameter of the spots which ranged from some eight to ten feet in the smaller ones to two to four rods in the larger (pl. IX, fig. 2). In no case have we had opportunity to examine the plants immediately following the lightning stroke, but plants taken a few days thereafter appear as follows. In the

mildest cases the injury occurs as a shrunken region about at the surface of the soil. The stem soon collapses here and the top falls over. Meanwhile the stem shrivels and browns progressively above and below this point, but fastest above (pl. IX, fig. 1). The pith in this region dies, browns and collapses, giving hollow stems but without rapid soft rot such as may occur with the bacterial black leg. Microscopic examination and cultures have failed to discover any bacterial or fungus organism considered parasitic. In the advanced cases with stems still living, the tissues are shrunken and collapsed for three-fourths of the length, from a short distance below the ground line nearly to the tip. Some of the partially injured stems immediately bordering the dead area gradually die, so that the spot tends to increase slowly in diameter for a week, but thereafter remains unchanged. No evidence of splitting or mechanical rupture of stems has been observed.

It will thus be evident that neither the individual plants nor the affected spot as a whole bears any close resemblance in appearance or development to any known potato disease. This, with the evidence which in at least three cases has coupled the inception of the injury with observed lightning strokes and the belief of numerous potato growers that lightning is the cause, has convinced us of the correctness of this interpretation.

It is only necessary to record further that these spots have been seen in Wisconsin on both sandy and clay loam fields, and that their distribution seems in no way dependent on the contour of the land. As we have observed them, they may occur in perfectly level fields, and in rolling fields they may appear at lower elevations as well as at higher.

Upon the publication of the abstract of this paper in the December issue of PHYTOPATHOLOGY, Professor F. C. Stewart wrote us that he had observed a couple of similar cases in New York potato fields last summer. These so closely corresponded to our description of lightning injury that he accepts this as the explanation of their occurrence. "In each case there was a single circular area in which the plants were mostly dead. The spots were 15 or 20 feet in diameter."

No similar injury to the other neighboring field crops has been observed by either author. Nor has conversation with colleagues brought out evidence of such except in the case of sugar beets. Miss V. W. Pool tells us that in Colorado the beet growers believe that lightning may kill sugar beets in spots. We have similar reports relative to lightning injury in tobacco fields. Mr. J. W. Brann, who is investigating the diseases of ginseng in northern Wisconsin, tells us of two cases of lightning injury to this crop which came under his observation. Ginseng is grown under artificial shade consisting of a lath screen supported on posts. In both of these cases the posts were struck and split by the stroke. In one case

it killed the aerial parts of the plants for a radius of some two feet about the bottom of the post, but the roots showed no apparent injury. In the other case it killed practically every top (aerial part) for a radius of ten feet about the post. Moreover, it killed practically all of the fibrous roots of these plants, but left the fleshy tap root with its terminal bud apparently unharmed. As indicating how different the result may be with different plants we have the statement of Mr. W. A. Orton that he saw a field at Blackshear, Georgia, where the line dividing a crop of cotton and corn (maize) passed through the center of a lightning-struck spot. On one side the cotton was killed over a semicircular area in the manner described in this article; across the line some of the corn stalks were split by the stroke, but no further injury was evident and none of the corn plants died. On the other hand, Dr. E. T. Bartholomew tells us that he has seen in a Kansas corn field a circular area something like two rods in diameter where all the plants had been killed, supposedly by a lightning stroke. It seems certain, however, that such lightning injuries to corn and grain crops cannot be as frequent as we have noted in potato and cotton fields else they would have become a matter of common knowledge among farmers. Of course, these fields are equally exposed to such electric discharges and if injury occurred it would be especially conspicuous in such crops. Meteorologists recognize the liability of lightning to strike in open fields and on comparatively level surfaces.[1] Dr. Bartholomew has told us farther of a Kansas observation where he saw lightning strike in a grass field, setting fire to dry grass. The land was undulating and rose to a gentle hill on one side of this field with woodland on the other. This corresponds with our observations in potato fields that such strokes show no marked preference for higher elevations or objects.

COMPARISONS WITH EUROPEAN RECORDS

We have found no reference to lightning injury to cotton, but Colladon[2] reports the effect of lightning injury to grapes which resembles the damage to cotton in some respects. These include the circular area affected, with greatest injury at the center, the slower death of some plants and the brick-red coloration of their leaves.

[1] See Abbe, Cleveland, Monthly Weather Rev. **32**: 323, 1904. Dr. Abbe recognizes the lesser liability of such a stroke upon a smooth plane surface as of still water, but points out how lightning may strike upon such irregularities as waves of water and minor projections from land surfaces, and cites evidence of such occurrences.

[2] Colladon, Daniel. Effets de la foudre sur les arbres et les plantes ligneuses Mém. de la soc. de phys. et d'histoire nat. de Genève **1872**: 548–553.

Rathay[3] has also noted the reddening of the leaves of lightning-struck plants and states that, in all authentic cases, it resembles the red coloration resulting from mechanical ringing of the branches, the injury in both cases being principally to the cambium layer.

Frank and Sorauer,[4] Colladon,[5] Appel,[6] and Spieckermann[7] all have published notes on lightning injury to potatoes.

Appel records that these evidences of lightning stroke appeared in spots including some rather broad areas. In these the plants in the middle were at once killed while nearer the margin of the affected area they were so torn and weakened as to become an easy prey to Fusarium. Dr. Appel in conversation further tells us that in one field he saw several such spots of which the larger was some fifteen feet in diameter. He has also seen local injuries to grapes due to lightning where an area of twice this size was involved. Similar cases of killing of sugar beets have come under his observation where instead of one large spot there would be three or four smaller spots.

Colladon also describes the lightning as killing potato plants in spots of which one was six meters in diameter. He notes evidences of physical injury to the stems and Steglich (quoted by Frank and Sorauer) describes these as split and torn with ragged wounds.

Colladon and Seelhorst[8] describe injuries from lightning strokes in beet fields. Seelhorst speaks of one case where the injury covered a circular area about fifteen meters in diameter. In the middle of this area the beets were entirely killed; in the outer parts the leaves of some of the plants were yellow or withered with intermingled plants showing slight injury. In the fleshy roots of the injured beets small cavities appeared, especially in the upper portions.

[3] Rathay, Emerich. Ueber eine merkwurdige durch den Blitz an Vitis vinifera hervorgerufene Erscheinung. Denkschr. d. math.-naturwiss. Klasse d. Kais. Akad. d. Wissensch. Wien. 1891.

[4] Frank und Sorauer. Jahresber. über die Thätigkeit des Sonderausschüsses für Pflanzenschutz. Jahrbuch der Deutsch. Landw.-Gesellschaft **7**: 208. 1892. (Record observations by Steglich.)

[5] Cited by Sorauer, P. Handb. d. Pflanzenkr. **1**: 495. 1909 (3rd Ed.).

[6] Appel, Otto. Mitt. a. d. Kaiserl. Biol. Anst. f. Land. u. Forstw. **5**: 19. 1907.

[7] Dr. O. Appel informs us that Spieckermann recently published briefly on this in Berichte d. Landkammer f. Westphalien. We have not been able to consult this, however, or verify the citation. This author has a brief note under the title, Kartoffelpest oder Blitzschlag. Prakt. Blätter f. Pfl'bau u. Pfl'schutz. **1907**: 103.

[8] Seelhorst, V. Rübenbeschädigung durch Blitz. D. Landw. Presse **31**: 515. 1904.

EXPLANATION OF THE PHENOMENA

The evidence as to the nature of the lightning injury to these herbaceous plants is too fragmentary to furnish ground for a satisfactory explanation of why it is more serious upon some crops than others. Evidently the conditions are quite different with these low succulent plants than obtain with lightning injury to trees. In the latter the current passes the full length of the plant through relatively resistant tissue and the injury may occur throughout the length of the trunk. With the potato and cotton the injury occurs chiefly at or near the ground line. It seems clear that the lightning does not strike the individual plants generally over the injured area or at least that the main force of the electric current does not pass through the length of their stems. Instead the evidence indicates that the discharge is from the surface of the soil. In conference with Dr. Lyman J. Briggs of the Office of Biophysical Investigations, Bureau of Plant Industry, we have formulated the following explanation as best in accord with our observations. When an electric storm breaks suddenly following a period of dry weather and the first rain wets the top soil, there remains a layer of dry earth between this wet surface and the moist soil underneath, which is a poor conductor of electricity. When the lightning strikes the wet surface soil, it disperses in all directions, horizontally and then downward into the earth, following lines of least resistance. The plant stems and roots with their abundant water content are better conductors than the layer of dry soil just mentioned and so the electrical current passes through them. The tissues may thus be variously injured or killed depending upon the amount of current passing through them. With the cotton, it seems that the higher water content of the bark and cambium may make these regions the especial paths thus explaining why the injury appears greater in these.[9]

The strength of the current, of course, diminishes the farther it gets from the center of the affected spot, and consequently the lessened injury at the margins of the area. In some cases apparently the discharge may be broken and "strike" in several spots, near together.

This explanation seems in general accord with the idea as to injuries to plants by lightning or other electrical current as set forth by Stone and Stahl.[10]

It still remains to ask why some plants, e.g., cotton, potatoes and beets, seem more liable to injury than others, e.g., grass, grains and corn. It is

[9] See Stone, G. E. and Chapman, G. H. Electrical resistance of trees. Mass. Agr. Exp. Sta. Rpt. **24**: 144. 1912. Also, Stone G. E., Electrical injuries to trees. Mass. Agr. Exp. Sta. Bul. 156. 1914.

[10] Stahl, Ernst. Die Blitzgefährdung die verschiedenen Baumarten. 1912. (Jena.)

possible that the amount of injury is proportioned to the degree of resistance shown by the tissues and that the potato and cotton tissues are less favorable conductors. This may perhaps be determined by experiments. Again it is conceivable that the character and distribution of aerial parts or root systems, respectively, may in the one class of plants be more favorable to gradual electric discharge as compared with the tendency to electric accumulation and resultant lightning stroke in the other class. The greater number, uniformity of distribution, and similar height of the leaf or stem apexes of corn, grains, and grasses seem to make this a tenable theory, since these numerous points would seem more favorable for such gradual discharge than would the aerial parts of potatoes, beets, or cotton.

EXPLANATION OF PLATE VIII

FIG. 1. Cotton field showing circular areas, about in the middle of the photograph, where plants were killed by lightning and weeds have grown.

FIG. 2. Cotton plant killed by lightning, leaves and bolls still clinging to the branches.

FIG. 3. Lightning killed cotton plant showing shrunken darkened portion below the callus ridge at *a*.

FIG. 4. Lightning struck cotton plant which died slowly, showing pronounced callus formation at *a* with splitting of the dead bark below and young sprout above.

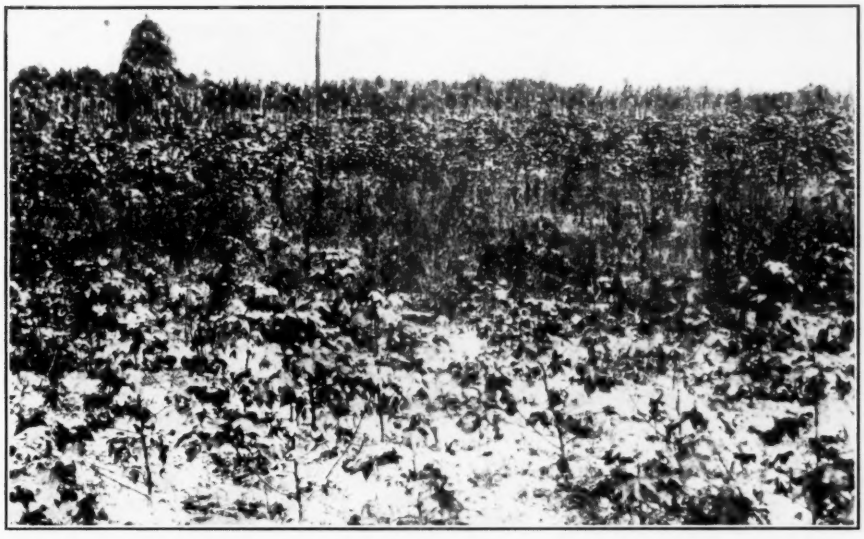

FIG. 1. Lightning injury in cotton field. (See explanation of plate.)

FIG. 2. Lightning killed cotton plant. FIGS. 3, 4 injured stems. (See explanation of plate.)

JONES AND GILBERT: LIGHTNING INJURY

Explanation of Plate IX

Fig. 1. Potato stem injured by lightning. From the margin of a lightning-killed area. It was evident that the initial injury occurred near the soil line, the roots being intact and the base of the stem alive and firm. The aerial portion of the stem was collapsed and black for two-thirds its length (a–a). The bases of the petioles and lateral branches (b, b) were also collapsed but the leaf blades were uninjured except as they were wilting through lack of water.

Fig. 2. Spot in a potato field, Barron County, Wisconsin, killed by lightning stroke. Note that this is on fairly level ground. Such injuries have been observed on perfectly level fields, on hillsides and in lower areas, with no evidences that topography influences location. Lightning struck on July 11, 1914, photographed in August by F. D. Otis.

FIG. 1. Potato stem injured by lightning. (See explanation of plate.)

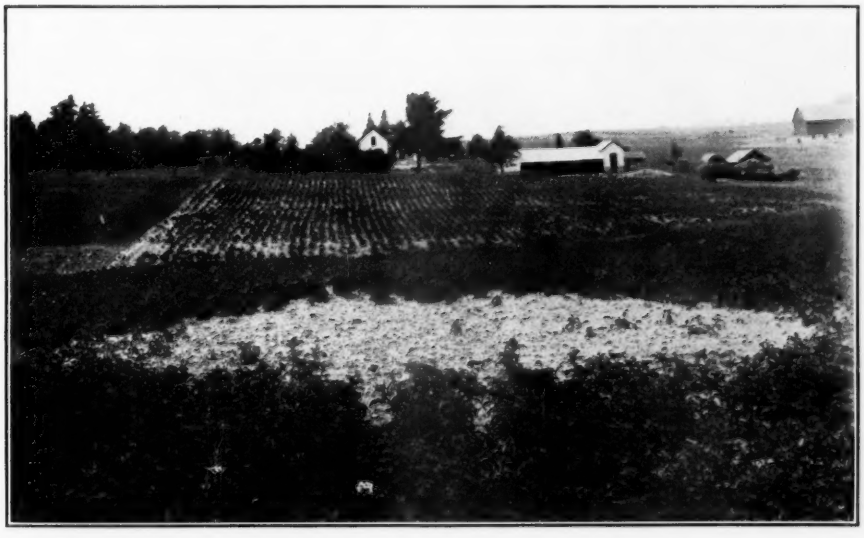

FIG. 2. Spot in potato field killed by lightning. (See explanation of plate.)

NEW LIGHT ON CURLY TOP OF THE SUGAR BEET

RALPH E. SMITH AND A. BONCQUET

WITH THREE FIGURES IN THE TEXT

The purpose of the present note is to record certain interesting facts which have been ascertained in a study now going on of curly top of the sugar beet.

Corroboration of relation of Eutettix tenella to Curly top. The evidence of Ball[1] (1909) and Shaw[2] (1910) as to this peculiar disease being incited by the sting of the minute leaf hopper, *Eutettix tenella* Baker, has never been fully accepted by entomologists and pathologists, owing to the unique importance of the fact and various points claimed to be lacking for absolute proof. Our work has, we believe, settled this question in the affirmative beyond any question. First we repeated the methods of Ball and Shaw, covering beet plants with gauze tents and cages, thereby confining the insect upon or excluding it from certain plants. This was done in the open field and also in the greenhouse, with plants grown from the first in insect-proof cages to exclude all but the individuals being experimented with. The results were uniformly in accord with those of the previous investigators. Curly top developed freely in plants attacked by this particular insect, and in no others. To further refine our methods sugar beet seed was planted in four-inch pots and two plants allowed to grow in each pot, keeping the whole in a Eutettix-proof cage from the first. After each plant had about 8 to 10 leaves, a single insect was confined in a vial upon one of the two plants in each pot, as shown in figure 1. In each case the plant thus infected showed the typical disease after the usual incubation period (about two weeks, in the greenhouse), while the other in the same pot did not.

In applying the insect for different lengths of time, it was found that as short a period as five minutes is sufficient to produce the disease.

A lesion and an organism in affected plants. All of the various gross symptoms and morphological types of this disease described by previous

[1] Ball, E. D. Leaf Hoppers of the sugar beet. U. S. Dept. Agr., Bur. Ent. Bul. 66, pt. iv. 1909.

[2] Shaw, H. B. The curly top of beets. U. S. Dept. Agr., Bur. Pl. Ind. Bul. 181. 1910.

writers (R. E. Smith[3] (1907), Townsend[4] (1908), Ball (l.c.), Shaw (l.c.)), together with some which they did not describe, are correlated with a specific interior lesion, always present in a greater or less degree in affected plants. This lesion is found in the phloem, in all parts of the plant where any visible, macroscopic irregularities appear, and to some extent where they do not appear, i.e., before their development. This effect consists at first in a denser-appearing condition of the cell contents of certain areas of sieve tubes and companion cells, followed by more or

FIG. 1. Curly top developed in one of two sugar beets in same pot, by application of *Eutettix tenella* in a vial for five minutes on one leaf.

less necrosis, collapse, discoloration, and often, in the end, development of new, irregularly formed, wound-healing cells in these areas. These lesions are sometimes scarcely more than small areas of slightly irregular cells in the phloem, while again they may be large discolored, necrotic pockets visible to the naked eye in the petioles and main veins as well

[3] Smith, Ralph E. Beet blight investigation. *In* Cal. Agr. Exp. Sta. Bul. 184: 240. 1907.

[4] Townsend, C. O. Curly top, a disease of the sugar beet. U. S. Dept. Agr., Bur. Pl. Ind. Bul. 122. 1908.

as in the roots. Figure 2 gives a diagrammatic idea of the location of these lesions in a vascular bundle of a large vein. They can be detected in lesser degree in the finest ramifications of the veins of curly top leaves.

From a long series of cultures it has been found that a certain specific bacterial organism appears to be a constant inhabitant of the tissues of sugar beets affected with curly top. This organism has been obtained very regularly from all parts of affected plants, even after the most severe surface sterilization with mercuric bichloride, and also by cutting out pieces of tissue with a flamed scalpel. More attention has thus far been devoted to the leaves than to the roots, and it is from these organs especially that the organism has been so regularly obtained by means of

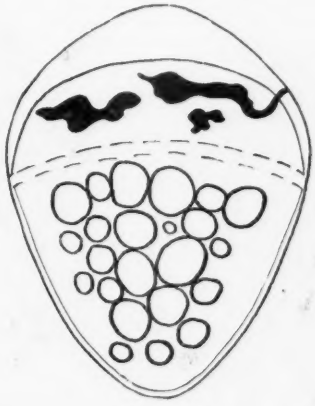

FIG. 2. Diagrammatic representation of location of lesions in vascular bundles of leaf veins.

cultures (by the ordinary bouillon tube isolation method). The leaf shown in figure 3 represents a typical case. In this leaf may be seen the visible indications of curly top in the lower three quarters of the left side and extreme lower right hand portion. After soaking for ten minutes in 1–1000 mercuric chloride solution in water and subsequent thorough washing in sterile, distilled water, bouillon tubes were inoculated with pieces of tissue from different parts of the leaf.

Pure cultures of the usual organism developed in the four tubes made from those portions of the leaf where the visible indications of the disease appear, while four tubes from the upper right hand half of the blade remained sterile. Numerous similar results were obtained in other cases. Here again the characteristic phloem lesion could be found underlying every swelling and irregularity seen on the surface (under) of the leaf.

The organism found agrees with *Bacillus dianthi* Bolley[5] (1896), originally described as the cause of the carnation disease afterward called Stigmonose. This organism has been found abundantly as a saprophyte

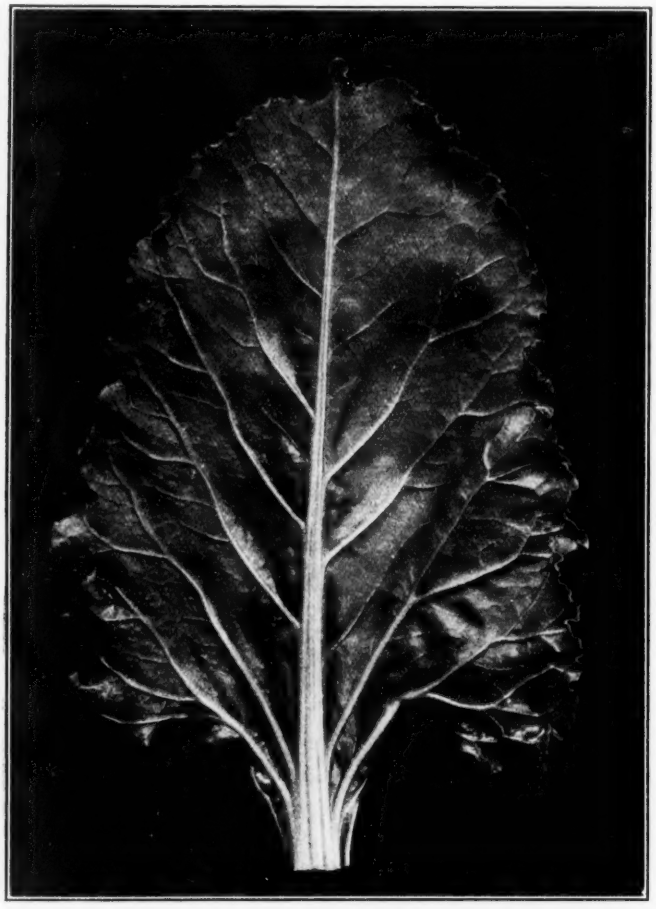

FIG. 3. Typical, partially-affected curly top leaf, from which Bacillus dianthi developed uniformly in cultures from left side, after severe disinfection, but not from right side. Phloem lesions also present in correlation with this.

in soil and on the surface of healthy beet leaves and is particularly abundant on the surface of beet seed. After surface sterilization, however, it disappears completely except in the case of curly top tissues. Beet

[5] Arthur, J. C. and Bolley, H. L. Bacteriosis of carnations. Ind. Agr. Exp. Sta. Bul. 59. 1896.

leaves sickly, yellow or partially dead from other causes do not show it at all under such conditions.

Curly top not produced by infection with B. dianthi. We have not succeeded in producing the disease by inoculating plants with this organism. We do not know whether it is the true inciting factor, causing and inhabiting the lesions described, a harmless invader, or a co-agent with some other factor.

Curly top transmissible by grafting. By grafting buds, connected with wedge-shaped pieces of root tissue, from diseased beets into the "shoulders" of healthy ones, we believe that we have produced the typical disease in each of the few instances thus far tried.

Microscopic detection of organisms in diseased tissues. Much difficulty has been encountered upon this subject. In the lesions above mentioned certain bodies of an apparently living nature are often detected, but whether they can be taken for *B. dianthi*, wholly or in part, or what their real nature may be, has not yet been decided. Further work is necessary upon this matter and upon the general subject of the specific nature of the effect of *Eutettix tenella* and the ontogeny of the disease.

UNIVERSITY OF CALIFORNIA
BERKELEY, CALIF.

SOME PROBLEMS OF PLANT PATHOLOGY IN REFERENCE TO TRANSPORTATION

F. L. Stevens

The purpose of this paper is not to present new facts but rather to suggest what is perhaps a new, or at least a little discussed viewpoint. The pathologist usually concerns himself with the occurrence of disease in the field or at the point of production, or with local storage troubles, much less frequently with questions as to what may happen to a plant product after it leaves the hands of the producer.

Questions of great importance may, however, arise concerning the changes which occur in transit, especially in the case of long hauls or of particularly perishable freight. Two or three illustrations may make the meaning clear and serve as the basis for further discussion.

Take the case, for example, of a carload of peaches originating in an orchard heavily infected with the monilia stage of *Sclerotinia fructigena*. Before packing all peaches known to be infected are discarded. The fruits to casual observation are sound when placed in the cars. When they arrive at their destination many show incipient or even well-developed rot. Again, a shipment of lettuce is made from a plantation infected with *Sclerotinia libertiana*. The plants visibly affected are sorted out. The shipment is badly affected at destination.

The product in both of these cases was infected when consigned. The history of the case and our knowledge of the spread and development of Monilia convince us that the peaches were diseased when shipped. In the case of the lettuce, since the fungus does not form conidia and, under these circumstances, may be spread only by contact, we may be sure that if infected at destination the shipment was also infected at the starting point. It appears clear to the writer that in both of these cases the responsibility rests with the shipper, just as much so as it would if a consignment of horses already infected with glanders but not yet showing the disease was placed upon the cars.

A case somewhat less clear might occur in a shipment of grapes which upon arrival is found to be badly affected with blue mold (Penicillium). We know that this fungus is a wound parasite, that it can gain entrance only through breaks in the skin. Such breaks may occur in picking, in packing, or may be due to the fact that the berries are over ripe, therefore

soft. On the other hand, they may be due to rough handling in the cars. The necessary spores are ever present, probably already upon the fruit, and yet no damage results without mechanical injury first. The responsibility clearly rests with the one permitting such mechanical injury.

Cases of an entirely different class are represented by the scab diseases of apple, potato, and peach. Each is of such slow development that it is inconceivable that even in very long slow hauls it could increase appreciably.

Other factors which enter into consideration are those of time and temperature. A consignment of asparagus or lettuce in prime condition if kept in a warm, humid atmosphere long enough, will rot in one or another way. In the language of the court they are "inherently liable to deterioration and decay."

The question of responsibility, therefore, will often hinge upon this "inherent susceptibility." How long under different temperature and humidity conditions will plant products remain wholesome? For present purposes, perhaps the following classification of diseases will help to clarify conceptions.

A. Slowly developing diseases which under no conditions will increase appreciably within a period of a few days.

Examples: Wheat smut, apple and peach scab, apple blotch and scab, Ascochyta of pea, potato scab, Fusarium and Phytophthora, tomato blossom end rot, watermelon anthracnose.

B. More or less rapidly developing diseases which can normally originate only at point of production.

a. Rapid:

Examples: Monilia of peach, various bacterial soft rots (?), plum and cherry Monilia, *Sclerotium Rolfsii*, canteloupe rots, *Sclerotinia libertiana* on lettuce, Botrytis on lettuce.

b. Less rapid:

Examples: Apple bitter rot, black rot, and pink rot, grape black rot, bean anthracnose, celery Septoria, egg plant ascochytose.

C. Rapidly developing diseases which can occur only on wounded plant parts.

Examples: Rhizopus on strawberry and sweet-potato, Penicillium on grapes, apples and oranges. Various bacterial rots, e.g., celery, asparagus, onion, et cetera.

D. Rapidly developing diseases which occur only on wilting or old products or under unsuitable condition of temperature or humidity.

Examples: Bacterial or mold infections of many kinds, Rhizopus, Penicillium, bacteria, et cetera.

Class A we understand well. All of the more important cases have probably been studied and described. We know also the general nature of classes B and C. The causal agents are well known but not the effects of temperature and humidity upon the rate of progress of the diseases. Class D is the one which presents the greatest inaccuracy of knowledge. Indeed, we possess as yet very little real knowledge which will enable us, for example, to say how old, under a given condition of temperature and humidity, a tomato may be without becoming liable to the decay which is its "inherent nature." Nor can we say with any accuracy how detrimental to keeping quality would be a change of a few degrees of temperature. Really accurate answers to such questions as these are of much importance, both because they will enable us to enlarge our power of control over these diseases and because the answers to the questions often involve determination of large financial responsibilities.

UNIVERSITY OF ILLINOIS

A SUGGESTION OF A NEW PHASE OF THE PROBLEM OF PHYSIOLOGICAL DISEASES OF PLANTS

CHAS. B. LIPMAN

The vagueness of the general term physiological plant diseases has of late been heightened by an increase in the number, diversity, and economic moment of manifestations which for want of a better classification have been relegated to that niche in the plant pathologist's cabinet. Despite the great volume of work which has been expended on that most troublesome group of plant ailments, the fundamental principles involved in their causation still remain shrouded in a thick veil of mystery. In an uncertain manner we ascribe their origin to some functional irregularity in the affected plant which may be induced by a variety of agents. Whether our methods of attack in attempting to solve these difficult problems have been ill advised or our conceptions with respect to them erroneous, it is not the purpose of this brief paper to discuss. Suffice it to say that the economic significance of "physiological diseases of plants" has in recent years assumed colossal proportions and scientific investigations with respect to them have contributed but little to their solution.

The foregoing observations on the present status of the subject under consideration have suggested to the writer other aspects of the problem which in turn hinted at new avenues of attack. Preliminary experiments based on such ideas, as explained below, indicated the existence of certain abnormal conditions in the soils upon which affected plants were growing which have since been found to obtain in many soils of California on which various physiological plant diseases occurred. It is only with a view to discussing these findings that these lines are written, in the hope that they may be pregnant with many suggestions for further work in a field which is certain to make grateful returns to the investigator sooner or later. The writer being, in a sense, a layman in the field to which his present readers largely belong, offers his observations in no spirit of criticism of past work, nor with any attempt at aspersion of such work and therefore ventures to hope that his observations will receive the consideration of patient thought, and his methods of a wider test than they are now receiving.

In another publication[1] the writer has pointed out that decidedly abnormal conditions, with respect to the nitrogen supply obtain in certain of our California soils upon which die-back (exanthema) and mottled leaf of citrus trees occur extensively. The abnormal conditions consist principally in a poor supply of available nitrogen but it is not infrequently found that the total nitrogen supply in such soils is very low. What is further of great interest is the fact that the meagre supply of available nitrogen referred to is not necessarily due to a lack of total nitrogen, but to incapacity on the part of the soil's flora, to render available such nitrogen as is present. In other words, the bacterial flora of a given soil, especially under the conditions of our arid valleys, may be capable only of rendering available the nitrogen contained in certain nitrogenous materials and not in others. Curiously enough the amount of nitrogen contained in a given material, as the writer will soon show more in detail elsewhere, does not appear to influence the degree of its transformation by the flora in question. Thus dried blood, a "high grade" nitrogenous fertilizer, may be worse than useless as a source of nitrogen to plants growing on the "abnormal" soil, if nitrifiability may be considered a criterion, whereas cottonseed meal or low grade tankage may have their nitrogen readily changed to nitrates by the various groups of nitrogen transforming bacteria present in that soil. Lest one might, however, gather from the latter statement that materials low in nitrogen are more readily attacked by the particular class of soil flora under discussion, it must be added that such flora are able to transform the nitrogen of materials of the highest grade into nitrates about as efficiently as the "low grade" materials just mentioned. Sulfate of ammonia for example, may be mentioned as such a material.

The foregoing statement merely gives a hasty sketch of the relation of certain "die-back" and "mottled leaf" soils, studied by us, to nitrogen as referred to in the paper above cited. Since those studies were carried out, however, we have found a similarly abnormal condition to exist widely in our interior valley soils on which such other physiological diseases as little leaf of the vine and little leaf of deciduous trees as well as general backwardness in growth are prevalent.

Based on the foregoing considerations the writer has offered the definite theory in lieu of, though in a sense consonant with, the vague one of malnutrition, that the physiological diseases mentioned above and perhaps others, are caused by an insufficient supply of available nitrogen, involving in some cases not only nitrogen hunger but internal disturbances in the cells owing to their growth in unbalanced solutions as media. The

[1] Science, n. ser. **39**: 728.

word theory is intended to serve in its most complete significance here since on the plant side the writer possesses in support of the idea, conclusively, only greenhouse experiments in pots, besides the laboratory investigations on the nitrogen content and powers of nitrification (for different materials) possessed by the abnormal soils under consideration. While field experiments in test of the theory are now proceeding no conclusive data have as yet been obtained.

That the general idea of malnutrition, with respect to nitrogen, of the affected plants in the cases of the diseases above named, may have many phases and the causative agents vary widely under different soil and climatic conditions, is indicated by the following possibilities in the direction of an insufficient supply of available nitrogen.

1. The total nitrogen supply may be very low and hence an insufficient supply of available nitrogen is almost certain to follow.

2. In soils in which sufficient nitrogen is supplied the form of the latter may render it unsuitable for transformation to nitrate by flora existent in that soil.

3. In soils high in carbonates, particularly those of the alkali earths, ammonia may be set free as rapidly as it is formed by the ammonifying bacteria and very little nitrate be produced in those soils as a result. In such cases nitrogen starvation would be expected.

4. A possibility which was suggested in the paper above cited is one which appears of lesser importance in the light of our recent results, and that is the toxic effect of ammonium compounds directly assimilated by plants in soils incapable of producing nitrates. In any event, of course, this could only occur in plants which are injured by nitrogen in the form of ammonia.

The conditions, in turn, which may contribute to those just named are numerous. So far as the total nitrogen supply is concerned it is well known to all soil chemists who have an intimate acquaintance with California soils that the latter taken by and large are very poorly supplied with that element. This is most strikingly the case in the lower Sacramento and the San Joaquin valleys and in the desert valleys of the state including the Imperial and Coachella valleys, and the San Bernardino region. The total nitrogen in very many of these soils may be found to be equal to 0.01 per cent or 0.02 per cent. It is only the great advantage of the soils' depth in the arid regions, taken as a whole, that makes possible the normal growth of some plants on such soils. In other words, a large internal soil surface with a small nitrogen content when open to root development serves in lieu of a small soil surface with a higher nitrogen content. If for any reason, however, the greater soil depth should not be available to root development by reason of a bad physical con-

dition, the presence of hardpan or other interference, it is obvious that the amount of nitrogen in the shallow surface soil layers available for root development, will soon become inadequate for the plant's support and trouble of some kind is bound to ensue.

In the event of a sufficient supply of total nitrogen on the other hand it is easy to conceive of soil conditions which could inhibit the transformation thereof into available form. In the first place as our experiments show, the soil flora may be such as to require nitrogen in certain forms in order to render it "available" or to transform it into nitrate. Any unsuitable form of nitrogen, no matter how good it may be from the chemical standpoint may not only fail to yield nitrates in the soil but may actually be instrumental in causing losses in the soils' original nitrate supply. In addition to such conditions, however, many other conditions may contribute to a soil's inefficiency in the direction of transforming organic nitrogen into nitrates. Poor drainage, bad physical conditions, acid reaction of the soil, excess of soluble salts of a certain nature, excess of difficultly decayed cellulose material, may all very seriously hinder the transformation of soil nitrogen to nitrates. In the case of a crop accustomed only to nitrate nitrogen or preferring it, such soil conditions must directly or indirectly operate to produce nitrogen starvation.

In connection with the last possibility above mentioned of a poor available nitrogen supply it must be stated that the field experience with die-back in citrus trees of Florida would seem to give it some support. In that state it has been often noted that the disease has been aggravated by the employment of nitrogenous fertilizers in certain organic forms. This can be explained on the basis of the loss of the soils' nitrate supply subsequent upon the introduction of say blood or high-grade tankage as above indicated. But it can also be considered as lending support to the idea of direct absorption of ammonia by the citrus tree (since ammonification of such organic nitrogen may proceed normally in those same soils), and thus causing disturbances in the tree growth through toxic effects as explained.

In brief, it would seem that a large variety of soil conditions due to the mode of a soil's formation or to effects following from certain methods of management might contribute to the circumstances above categorically enumerated. Field observations as well as the laboratory and greenhouse experiments described in this paper are almost wholly in support of the theory herein set forth. It is further a significant fact and one strengthening the foregoing ideas, that in soils poor in nitrogen the diseases named manifest themselves after two or more seasons subsequent to planting indicating an assimilation and depletion of the nitrate nitrogen stored in the soil (in many cases a virgin one) to the point at which nor-

mal plant growth can no longer continue. In the possibility of toxic effects from direct absorption of ammonia we should also expect the manifestation thereof to come after two or more seasons as it appears to do in the case of die-back in lemons.

In the greenhouse experiments mentioned in support of the ideas above enunciated, we have found it possible to produce normal growth of orange seedlings in soils from "die-back" areas only by the application of materials which are readily nitrified by those soils, e.g., sulfate of ammonia, carbonate of ammonia, and chloride of ammonia. Good results were also obtained with sodium nitrate, but the effect was not nearly as striking as in the case of the ammonium compounds just mentioned. During the term of these experiments which are still in progress, orange seedlings grown on the untreated soil have made little or no growth, despite the fact that they have been exposed for about two years to the dust-laden atmosphere of the greenhouse which could be reasonably expected to change the soil's biological nature.

In the case of grain, also, a relationship between the soil's nitrogen supply and normal growth has been observed by us which is very similar to that above described for various fruits. A more detailed discussion of that subject is given in a paper which is soon to appear.[2] In a paper by Brooks which appeared in this JOURNAL[3] recently similar causes to those here discussed are hinted at as being concerned in the production of blossom end-rot in tomatoes.

Pased very largely, therefore, on studies concerned with the total nitrogen content of soils and with the variation of the latter in power to nitrify different forms of nitrogen, and in a minor way on the greenhouse studies above described, the writer suggests the possibility of curing the diseases above described by direct or indirect soil treatment (depending on the conditions above indicated) of a nature to insure a good supply of available nitrogen. In soils poor in humus it would appear to be best to employ sulfate of ammonia, or cottonseed meal, while in soils rich in humus dried blood and high grade tankage are likely to give better results. It is not expected, moreover, that such treatment in the field is likely to be followed by quick improvement in the affected plants. The reason for this is that a tree root or vine root covers a very large area of soil and it is not possible to influence that large foraging surface of the plant's roots quickly. Nevertheless, large applications of the materials named, along with a good water supply and thorough tillage to insure the maximum degree of distribution of the nitrogenous materials should go far to assist and hasten the process of the plant's recovery.

[2] Proc. Soc. Prom. Agr. Science, 1914.

[3] Phytopathology **4**: 345.

One more point deserves consideration in this brief general statement of the writer's suggestion regarding the causative agents of some physiological diseases of plants. The question may naturally arise as to how one advancing the theory above briefly considered could account for good individual plants or groups of such which are, frequently, perhaps generally, found in the diseased areas. In other words, on the assumption that the soil is all alike in a given area we should expect to have all plant growth alike. This argument however is erroneous since there is no uniformity in soils to begin with, but even if this is set aside as of minor importance here, it must be remembered that individuals among plants as well as animals, are frequently more resistant than their fellows to untoward conditions. If in a given soil with a deficient supply of available nitrogen one plant should develop a much larger root system than its neighbor, it is obvious that it would have at its disposal a much larger amount of plant food since its roots would cover so much more internal soil surface. It is therefore not a valid argument against the theory here promulgated that the soil near good plants as well as that near bad plants in a field is poorly supplied with nitrogen in an available form. Such a fact would only indicate that the condition of the whole field was bad and that good plants are produced in parts thereof in spite of conditions and not because of them, through individual plant powers to obtain the necessary nourishment which are not possessed by plants in general in that same field. In cases, however, in which the soil surrounding the good plant does differ in nature from that of the poor plant in the same field, explanations for the differences can be easily given as indicated in one of the papers above cited.[4]

I wish to reiterate here that I am merely proposing as a justified theoretical consideration to connect certain very troublesome functional troubles in plants known as cases of malnutrition or as physiological diseases with a definite lack of a specific plant food element—nitrogen—in available or usable form. I do not desire to be understood as attempting dogmatically to explain the unexplained, nor, to lay claim in any sense to finality in my statements. Despite all that, the observed facts as above described are very striking, and the experimental results thus far obtained most encouraging. It is the writer's earnest hope that those interested in plant nutrition problems will at least give his theory the courtesy of a carefully controlled test and thus help us to arrive sooner at a proper understanding of its validity and practical significance.

UNIVERSITY OF CALIFORNIA
BERKELEY, CALIF.

[4] Proc. Soc. Prom. Agr. Science, 1914.

THE RÔLE OF SUCKING INSECTS IN THE DISSEMINATION OF FIRE BLIGHT BACTERIA

V. B. Stewart and M. D. Leonard

Since the experiments conducted by Waite[1] who demonstrated that bees are important in the spread of blossom blight, various agents have been associated with the dissemination of the fire blight organism, *Bacillus amylovorus* (Bur.) Trev. It has been shown by Whetzel[2] that man himself is often an active agent in spreading the disease and it is believed also that birds frequently carry the bacteria.

Without question all insects which occur on host plants susceptible to blight are capable of carrying the causal organism, though it is probable that many of these species are not instrumental in producing new infections. The very nature of the method of introduction of the bacteria precludes the possibility of this. Any insect that comes in contact with the germ-laden, gummy exudation of the blight disease may, no doubt, carry the bacteria to other trees, but the possibility of infection taking place depends upon the introduction of the causal organism into the host tissue. Repeated experiments have shown conclusively that except for blossom blight, the bacteria gain entrance and produce an infection in the host tissue only through a wound or injury. From this fact it is therefore natural to infer that insects which feed by sucking the plant juices are more likely to spread the disease. They visit exuding blight lesions, become smeared with the gummy substance and carry the infection material to other shoots. Here some of the bacteria are deposited in the feeding punctures made by the insects and produce new blight infections. Insects which do not puncture the tissues in feeding are perhaps less apt to spread twig blight. They are of importance only in carrying the fire blight bacteria to wounds in the tissue such as mechanical injuries and the like.

The experiments and observations made by the senior writer[3] show that the tarnished plant-bug (*Lygus pratensis* L.) is capable of carrying

[1] Waite, M. B. Life history and characteristics of the pear blight germ. Proc. Amer. Assoc. Adv. Sci. **47**: 427–428. 1898.

[2] Whetzel, H. H. The blight canker of apple trees. New York (Cornell) Agr. Exp. Sta. Bul. 236: 104–138. 1906.

[3] Stewart, V. B. The importance of the tarnished plant-bug in the dissemination of fire blight in nursery stock. Phytopath. **3**: 273–276. 1913.

the causal organism to healthy shoots where the bacteria gain entrance to the host tissue through the feeding punctures made by the insect. Forbes[4] was probably the first writer to attribute to the tarnished plant-bug the capability of spreading the fire blight disease. He says, "There is considerable reason to suppose that these insects are often active agents in conveying the virus of the blight of the pear and apple from tree to tree by inoculation with the infected sap."

The constant appearance of other sucking bugs during the growing season and their association with outbreaks of the fire blight in the nursery, prompted further experimental work by the writers in order to determine whether any of these insects were capable of producing fire blight infections. For the experiments such species, occurring on apple stock, were selected as could be collected most easily in sufficient numbers and which also were believed to be most important in transmitting the fire blight organism. The names of the species are as follows: *Adelphocoris rapidus* Say, *Campylomma verbasci* Herrick-Schaeffer, *Orthotylus flavosparsus* Sahlberg and *Poeciloscytus basalis* Reuter.

Young apple seedlings with succulent shoots were also selected and the insects for the experiments were obtained from weeds at some distance from the nursery plantings rather than from the nursery trees themselves. This was done in order to eliminate as far as possible, any chance of using insects already smeared with fire blight bacteria due to their having visited diseased trees. Cylindrical cages, about 15 inches in height and 4 inches in diameter, made from wire-cloth, 12 meshes to the inch and covered with cheese-cloth, were used to cage the bugs over each tree. The bugs were transferred to the cages and the cages inverted over the trees, care being taken not to injure the shoots during the operation. A six-days-old, pure, agar culture of *Bacillus amylovorus* was employed for all experiments, the culture being smeared on the tips of the shoots by means of a camel's hair brush.

EXPERIMENT 1

Campylomma verbasci Herrick-Schaeffer. Length, about 3 mm.; upper surface greenish gray or a dirty grayish white; thorax often tinged in the middle with darker; antennae with two blackish rings near base; legs spotted with black; underside of abdomen brownish or black. Breeds commonly on the mullen, *Verbascum thapsus*.

The experiment was allowed to run from July 7 until July 22, 1914, when the final results were recorded. In two of the cages some of the bugs remained alive throughout the first seven days of the experiment.

[4] Forbes, S. A. The tarnished plant-bug. Farmer's Review, Feb. 28, p. 150, 1884.

The tips of the six shoots of three trees were smeared with the agar culture of *Bacillus amylovorus* and ten specimens of *Campylomma verbasci* caged over each tree. When the cages were finally removed four of the six shoots were badly blighted; the failure of the other two shoots to become diseased was attributed to their hard and woody condition, rendering them more resistant to the disease.

Ten bugs were allowed to run over an agar culture of the causal organism for one hour and then caged over a tree with four shoots. Two weeks later three of the shoots were blighted.

The eight shoots of three trees were smeared with the culture, but the bugs were excluded from the cages placed over the trees in order to protect them from other insects. None of the shoots blighted on these check trees.

Ten bugs were caged over a tree with three shoots, but the culture was not smeared on the tree or on the bugs. All of the twigs were healthy and free from injury two weeks later.

EXPERIMENT 2

Orthotylus flavosparsus Sahlberg. Length, about 3 mm. This little insect may be readily recognized by the fact that it is light green in color and the whole body is sprinkled with white. It is found commonly throughout the summer months in the region of Ithaca, New York, on *Chenopodium album*.

This experiment was set up July 22 and final notes taken August 3, 1914. The six shoots of three trees were smeared with a culture of *Bacillus amylovorus* and fifteen specimens of *Orthotylus flavosparsus* caged over each tree. Two weeks later all six shoots were badly blighted.

Fifteen bugs were caged over a tree with three shoots but the culture was excluded. All of the trees remained healthy.

The seven shoots on three trees were smeared with the agar culture, but the bugs were excluded from the trees by means of the cages. None of the shoots were blighted when the final observations were made August 3.

EXPERIMENT 3

Poeciloscytus basalis Reuter. This Capsid is very similar in appearance to the lighter colored specimens of the tarnished plant-bug. It is slightly smaller, however, being about 5 mm. in length; the yellowish mark on the scutellum is never Y-shaped or broken into three distinct spots, but is wedge-shaped, narrowing posteriorly. Instead of a yellowish spot on the wings there is a reddish or orange marking.

The following experiment was started July 29, 1914, and completed August 11. The seven shoots of three trees were smeared with an agar culture of *Bacillus amylovorus* and about twelve specimens of *Poecilocytus basalis* caged over each tree. The final observations were made August 11 when four of the seven shoots were blighted. Owing to the continued dry weather, the shoots made but little growth throughout the experiment. Those which blighted did not suffer so severely as the shoots in previous experiments, due to the hard and woody condition of the tissue which rendered them more or less resistant to the active progress of the disease.

Several of these insects were allowed to run over an agar culture of the causal organism for twenty minutes after which ten of them were caged over each of two trees. Two weeks later five of the seven shoots on the two trees were badly blighted.

Seven shoots on three trees were smeared with the agar culture and the insects excluded. Cages were placed over the trees to protect them from the attacks of other insects. All of the shoots remained healthy.

Ten bugs were caged over a tree with three tender shoots and the culture was excluded. At the conclusion of the experiment, the shoots were free from disease. Two of the shoots showed a slight discoloration, probably due to the feeding punctures of the bugs, but the injury did not develop any further and the trees remained healthy.

In several of the cages, some of the bugs were still alive at the close of the experiment.

EXPERIMENT 4

Adelphocoris rapidus Say. Length, 5 to 6 mm.; head and front margin of thorax reddish brown, remainder of thorax yellowish, with two large dark brown or blackish spots, which are sometimes confluent, thus forming a transverse band; wings brownish, bordered on the outside with yellow and with a reddish spot about two-thirds the distance back; antennae yellow, alternately banded with dark brown or red; eyes dusky; legs reddish brown; feet dusky at tip.

The experiment was set up July 29 and completed August 11, 1914. The three shoots of one tree were smeared with an agar culture of *Bacillus amylovorus* and nine nymphs (stages 3, 4 and 5) and one adult were caged over the tree. Two weeks later all these shoots were badly blighted.

Ten nymphs (in several stages) were caged over a tree with three shoots which had been smeared with the culture. After ten days the cage was removed and all the shoots were blighted.

Four bugs (two nymphs and two adults) were caged over a tree with two shoots, but the bacteria were excluded. After a period of two weeks

both shoots were free from disease and no injury was apparent. One of the adults was still alive when the cage was removed.

As a check on this experiment, the ten shoots on four trees were smeared with the agar culture of the organism and the bugs were excluded. Cages were placed over the trees to protect them from the attacks of other insects. All of the trees remained healthy.

From the results of the experiments conducted it is apparent that all of the above named species are capable of producing fire blight inoculations when the causal organism is present, and are undoubtedly instrumental in spreading the disease.

Where the insects were allowed to run over the agar culture of *Bacillus amylovorus* and then transferred to the trees, nearly all of the succulent shoots blighted. Also a large percentage of the shoots became diseased when smeared with the agar culture and the bugs caged over them. The insects attacked the tender tips of the shoots and produced lesions in the tissue which afforded an entrance for the blight bacteria. In all cases where either the agar culture of the causal organism or the bugs were excluded from the trees, none of the shoots blighted. It therefore seems natural to infer that the appearance of these sucking bugs on the trees is of extreme importance, especially when conditions are favorable for the development of the fire blight; their presence on the diseased trees should increase the spread of the causal organism.

Without question the tarnished plant-bug is worthy of special consideration as a fire blight disease disseminator and also the species *Campylomma verbasci* is undoubtedly of particular importance. This is due to their great abundance in the nursery blocks and to the fact that their attacks are chiefly confined to the tips of the shoots. These two species are frequently found in great numbers, as many as ten or twelve having been observed on a single nursery tree. Where there has been sufficient rainfall and cultivation to cause a rapid and succulent growth of the trees, the presence of sucking bugs on diseased shoots affords conditions favorable for the wide dissemination of the bacteria.

On the other hand, it is to be noted that sucking bugs may be present in great numbers without the occurrence of much blight. This may be attributed to two factors: the absence of hold-over cankers to furnish a source for blight infections and, what is of more importance, the unfavorable condition of the trees for infections to occur. This was especially emphasized during the season of 1914. In one large nursery, throughout the months of July and August, there was abundant rainfall, causing the trees to make a rapid growth. Considerable blossom blight appeared in a large orchard nearby and on being spread to the young nursery stock, a severe outbreak of the disease occurred and thousands of trees were

destroyed. In another large nursery located in a different section of the state where the rainfall was extremely light, practically no blight was observed throughout the summer, although the conditions were in many respects the same as those in the other nursery. A large amount of blossom blight was present in adjoining orchard trees; the stock had received about the same amount of cultivation and sucking bugs were present in great numbers; but the long-continued drought checked the growth of the young shoots, causing them to be not only more resistant to the attacks of the blight bacteria, but also not so liable to injury by these insects.

Because of the seasonal variations in the numbers of sucking insects which are capable of spreading fire blight in the nursery blocks, it is impossible to say which of several species are of most importance in blight dissemination.

Practically no experimental data are at hand with reference to the spread of twig blight in orchard trees and the writers are of the opinion that this question is of special interest. Indications point to the fact that certain sucking insects disseminate the bacteria in the orchard, but this has not been definitely determined. The well known apple red-bugs, *Heterocordylus malinus* Reuter and *Lygidea mendax* Reuter, which often so seriously infest orchard trees[5] are certainly able to spread the blight, as they puncture the tissue at the tips of the young twigs thus affording an entrance for the blight bacteria. From all indications the same species may also increase the amount of fruit blight which is sometimes common in certain varieties of apples. This may be particularly true where fruit blight is so abundant in the early part of the summer. Several specimens of fruit blight were received during the autumn of 1914 from various apple orchards in New York State and frequently the blighted apples showed red-bug injury. In the month of September, 1914, the writers visited a badly blighted Tolman Sweet apple tree which had been infested with red-bugs and a considerable portion of the fruit was blighted. Twig blight was abundant on the tree and many of the lesions were exuding the gummy substance characteristic of fire blight infections. There is some question as to whether red-bugs were responsible for the large amount of fruit blight as but few of these insects occur on trees after the latter part of July. On the other hand, undoubtedly other closely related species were present. These insects visiting the twigs, became smeared with the sticky ooze of the blight and carried the bacteria to the fruit, the organism gaining an entrance through the feeding punctures of the bugs.

[5] Crosby, C. R. The apple red-bugs. New York (Cornell) Agr. Exp. Sta. Bul. 291: 211-225. 1911.

Of particular interest is the recent work by Parrott and Hodgkiss[6] on the false tarnished plant-bug (*Lygus invitus* Say) as a pear pest. They report this insect, which is closely related to the tarnished plant-bug, as causing severe injury to the fruit of orchard pear trees. Doubtless the false tarnished plant-bug is capable of transmitting the blight bacteria and probably is of considerable importance in the spread of the disease. These writers also mention *Campylomma verbasci* Herrick-Schaeffer which is so common in the nursery, as also occurring on orchard pear trees. They report *Paracalocoris colon* Say, also closely related to the tarnished plant-bug, and the false red-bug, *Lygidea mendax* Reuter as feeding on the succulent growth of orchard pear trees.

Certain species of flies are attracted in considerable numbers to the gummy exudations of blighted trees and they, no doubt, carry the bacteria from place to place. It is possible that these flies may produce fire blight inoculations by injuring the tissue with their claws. However no experimental data are at hand to substantiate this point.

In view of the experimental work which has been done relative to the dissemination of fire blight bacteria in nursery stock, it seems desirable that similar studies be made on orchard trees in order to determine if possible, which agents are of most importance in the spread of this disease, particularly of twig blight.

Meanwhile cooperative experiments between pathologists and entomologists, which will lead to the control of these sucking bugs, should be encouraged by all interested in holding the disease in check. These species of insects, not only cause considerable injury to the trees, but from the nurserymen's standpoint, at least, the control of sucking insects would undoubtedly be a determining factor in reducing the annual losses caused by the fire blight disease.

DEPARTMENTS OF PLANT PATHOLOGY AND ENTOMOLOGY
CORNELL UNIVERSITY
ITHACA, NEW YORK

[6] Parrott, P. J. and Hodgkiss, H. E. The false tarnished plant-bug as a pear pest. New York (Geneva) Agr. Exp. Sta. Bul. 368: 363–384. 1913.

NOTES ON THE DISTRIBUTION AND PREVALENCE OF THREE IMPORTANT SWEET POTATO DISEASES

L. L. Harter

STEM ROT

In 1914, Harter and Field[1] published investigations on the sweet potato stem rot in which it was shown that this disease was caused by *Fusarium hyperoxysporum* Wr. and *F. batatatis* Wr. It was pointed out among other things that the disease had been studied in plants collected in New Jersey, Delaware, Maryland, Virginia and Alabama, and the causal organisms isolated. Likewise attention was called to the fact that different pathologists had reported the disease in other states, and that these investigators, without apparently making a thorough study of the disease, had followed the conclusions of Halsted, who claimed *Nectria ipomoeae* to be the cause of the trouble. However, after it was shown that this fungus was not the true cause of the stem rot, the distribution of the disease as determined by the presence of *Nectria ipomoeae* on plants was questionable. Therefore to determine with certainty the distribution of this and other important sweet potato diseases, the writer personally visited a number of sweet potato growing sections in the different states during the fall of 1914. In addition to the states already mentioned, the disease was found in Ohio, Illinois, Missouri, Iowa, Kansas, Oklahoma, Arkansas, North Carolina, Georgia and Mississippi. From these data it is seen that the stem rot has already a wide range of distribution.

At Marietta, Ohio, and Cobden and Anna, Illinois, stem rot was found on the Yellow Jersey variety and the total loss was estimated at from 10 to 40 per cent. The farmers claimed that the disease was not known until seed potatoes had been imported from New Jersey, Delaware and Virginia. The disease was found at Sikeston, Missouri, but only to a limited extent, the loss to the crop not exceeding more than 5 per cent. At Muscatine, Iowa, the Yellow Jersey is the principal variety grown and in many fields 50 to 60 per cent of the plants were infected with stem rot. In fact, so destructive has the disease become to this variety that a whole crop is sometimes lost. It is interesting to note in this connection that the Southern Queen variety, growing beside the Yellow Jersey under identical conditions, was unaffected.

[1] Harter, L. L. and Field, Ethel C. The stem rot of the sweet potato (Ipomoea batatas). Phytopath. **4**: 279–304, plates 14–16. 1914.

Stem rot was found at three points in Kansas, namely, Wamego, Manhattan and Herington. Sweet potatoes are grown only to a limited extent at Manhattan and Herington, but at Wamego this crop forms an important industry. The Yellow Jersey variety is the one most generally grown and in some fields the stem rot destroys as much as 60 per cent of the crop. In both Iowa and Kansas the farmers claimed that the disease had been introduced by means of seed imported from eastern states. At Oklahoma City stem rot was fairly common in the Yellow Jersey, Pumpkin Yam and Bradley Yam varieties. This being a new section for sweet potatoes, the loss was relatively small. At Fort Smith and Lincoln, Arkansas, stem rot was very common in the Nancy Hall and Yellow Jersey varieties. No cases of the disease were found in Yellow Yam, a variety grown very extensively in Arkansas, Texas and Oklahoma.

In North Carolina, Georgia, Alabama and Mississippi, the other states where stem rot was found, Yellow Jerseys are not grown, but Nancy Hall, a variety equally susceptible to the disease, is widely cultivated. In this variety the disease was found at Goldsboro, North Carolina, Savannah, Georgia, Hattiesburg, Mississippi, and at Foley, Bay Minette, and Axis, Alabama. Only in Alabama was the disease observed to be causing any great loss. In this region it destroyed 20 to 50 per cent of the Nancy Halls and was found prevalent in the Porto Rican and Providence varieties. No stem rot was found in the Triumph, a variety grown extensively in the same region.

Fusarium batatatis was isolated from specimens collected at Foley, Alabama and at Savannah, Georgia, while at all other places *F. hyperoxysporum* was found to be the causal organism.

BLACK ROT

This disease caused by *Sphaeronema fimbriatum* (Ellis and Hals.) Sacc., has been found on specimens collected from many places in New Jersey, Delaware, Maryland and Virginia, at Marietta, Ohio, Cobden, Quincy, Epworth and Anna, Illinois; Sikeston, Missouri; Muscatine Iowa; Manhattan and Wamego, Kansas; Oklahoma City and Ada, Oklahoma; Fort Worth and Gainesville, Texas; Fort Smith, Lincoln and Little Rock, Arkansas; Goldsboro, Stacey, Salisbury and Newton, North Carolina; Savannah and Jesup, Georgia; Bay Minette, Axis, Madison and Foley, Alabama. The disease has therefore quite a wide range of distribution and it is probable that it occurs wherever sweet potatoes are grown. At many of these places the disease had caused heavy losses in the hotbeds and in the storage houses. In some instances the farmers regarded the black rot as even more serious than the stem rot.

FOOT ROT

This comparatively new disease (*Plenodomus destruens* Harter) has been previously reported[2] from Virginia and Sikeston, Missouri. It was found again in considerable abundance during the fall of 1914 at Sikeston, Missouri, and for the first time at Muscatine, Iowa, and at Marietta, Ohio. From the losses caused by this disease it is not unlikely that it will become a real menace to the growing of the sweet potato crop. At Marietta, Ohio, 75 per cent of the plants in a field of General Grant Vineless were diseased. At Muscatine, Iowa, foot rot was more generally prevalent and caused, on the whole, more loss, although no fields were found with such a high percentage of infected plants as in Ohio. At Sikeston, Missouri, the total loss to the crop was small but individual plants were found in which the organism had grown down from the stem into the roots, forming fruiting bodies and partially decaying the tissue. A pure culture of Plenodomus was secured from decayed tissue within the potato. This organism was found to cause a serious hotbed trouble in Virginia during the spring of 1914, where it occurred in abundance on the young plants in the field taken from the hotbeds.

U. S. DEPARTMENT OF AGRICULTURE
BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

[2] Harter, L. L. The foot rot of the sweet potato. Jour. Agr. Research **1**: 251–273, plates 23–28. 1913.

PHYTOPATHOLOGICAL NOTES

Personals. Dr. P. J. Anderson, formerly Field Pathologist with the Pennsylvania Commission for the Investigation and Control of the Chestnut Blight Disease, has been appointed Instructor in Botany at the Massachusetts Agricultural College.

Dr. Otto Appel has accepted a temporary appointment in the Bureau of Chemistry of the United States Department of Agriculture, to advise regarding the industrial uses of potatoes.

Errata. The following errata were made in the article The Pathological Histology of the Endothia Canker of Chestnut in PHYTOPATHOLOGY, volume 4, June, 1914:

The description given under figure 2 should have appeared under figure 1; the description given under figure 3 should have appeared under figure 2; the description given under figure 1 should have appeared under figure 3.

Mordecay Cubitt Cooke. The death is announced of the veteran Mycologist, Doctor M. C. Cooke, which took place on November 12, 1914 at East Southsea, England, at the ripe age of nearly 90 years. Doctor Cooke was born at Horning, Norfolk, England on July 12, 1825. A biographical sketch will appear in the first number of our Journal for 1916.

REPORT OF THE SIXTH ANNUAL MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The sixth annual meeting of the Society was held in the Medical Building of the University of Pennsylvania, Philadelphia, Pennsylvania, December 29, 1914, to January 1, 1915, in connection with the American Association for the Advancement of Science.

About ninety-five members were present and a program of fifty-eight papers was presented. The abstracts of these papers were published in the December number of PHYTOPATHOLOGY. Several new members were elected making a total of 293.

Joint sessions were held with Section G of the American Association for the Advancement of Science and with the Botanical Society of America.

The following officers were elected for 1915:

President, H. H. Whetzel, Cornell University, Ithaca, N. Y.

Vice-President, W. A. Orton, U. S. Department of Agriculture, Washington, D. C.

Councilor for three years, Mel T. Cook, New Brunswick, N. J.

Editorial Board, Donald Reddick was elected chairman of the Board of Editors for three years.

The following associate editors were elected for three years: H. W. Barre, E. A. Bessey, H. R. Fulton, W. T. Horne.

C. L. Shear was elected business manager for the coming year.

The Society decided to hold its next annual meeting at Columbus, Ohio, in connection with the meeting of the American Association for the Advancement of Science. The Society also voted to hold a special meeting at San Francisco, California, August 2 to 7, in connection with the meeting of the American Association, and the secretary was instructed to arrange with the secretary of the Western Branch of the Society the necessary details in connection with the program.

The following recommendation in regard to the attendance of the secretary at the special meeting was adopted:

"In view of the present state of the treasury and the unusually heavy traveling expenses involved, we do not now authorize the attendance of our secretary at the San Francisco meeting at the Society's expense.

"This action is not to be construed as restricting the freedom of the Council to authorize this journey if unforeseen conditions make it urgently important."

REPORTS OF COMMITTEES

The Committee on Affiliation and Relation to other Societies presented the following report which was accepted and the Committee discharged:

Appreciating highly the cordial spirit shown in the proposition of the Committee on Affiliation of the Botanical Society of America, and the courteous manner in which this has been presented, we reply as follows: In view of certain financial and other obligations resting upon this Society we consider it inexpedient to urge further affiliation of the two societies at present.

We trust, however, that this may not be construed as indicating any unwillingness on the part of the American Phytopathological Society to give further consideration to this question.

F. C. STEWART,
C. L. SHEAR,
Committee

The Committee on Common Names of Plant Diseases presented a report suggesting ten rules to be followed in the preparation of a list of common names of plant diseases. The Committee also recommended that the Society appoint a permanent committee of five on common and scientific names.

The Society voted that the important progress represented by the report of the Committee be recognized and that the Committee be continued and requested to submit a copy of its recommendations to each member of the Society for suggestion and criticism.

The Committee on Ways and Means, consisting of F. C. Stewart, C. L. Shear, Donald Reddick, H. H. Whetzel, and C. E. Bessey, made the following report, which was adopted and the Committee continued:

The Committee has tried to interest certain wealthy persons in providing an endowment for PHYTOPATHOLOGY, but, so far, has failed. However, the first $50 towards a permanent fund has been contributed by Dr. W. G. Farlow. It is recommended that the Secretary be instructed to thank Dr. Farlow for this gift.

It also recommends that the Society proceed to incorporate under the laws of the District of Columbia.

Upon motion, the Society authorized the secretary-treasurer to select the two other persons necessary and to proceed to secure articles of incorporation for the Society under the laws of the District of Columbia.

The Committee on Ways and Means was empowered to appoint a sub-committee to confer with Dr. Flexner, of the Rockefeller Institute for Medical Research, regarding plans for the promotion of phytopathology. L. R. Jones was appointed chairman of such sub-committee, with power to select his associates.

The report of the Committee on Bibliography was presented by the chairman, L. R. Jones. The results of the Committee's efforts are to be found in the lists of literature now appearing regularly in PHYTOPATHOLOGY. Great credit is due Miss E. R. Oberly, Librarian of the Bureau of Plant Industry, and the Chief of the Bureau of Plant Industry, Dr. W. A. Taylor, for their approval and assistance in this undertaking. The Committee was continued with instructions to consider the feasibility of issuing the list on cards for sale at cost.

Donald Reddick, chairman of the Committee on Business Arrangement with Williams and Wilkins Company, publishers of PHYTOPATHOLOGY, reported that the present contract with the company was very satisfactory with the exception that the returns from advertising had not reached the expectations of the Company and the guarantee should be reduced from $240 to $150 for the coming year. It was recommended that the present arrangement with Williams and Wilkins Company, with this modification, be approved for the coming year. The report and recommendations were adopted.

The Committee consisting of G. G. Hedgcock, N. J. Giddings, and N. E. Stevens, appointed to audit the treasurer's account, reported that the accounts had been examined and found to be correct, and the report was adopted.

The treasurer's report was as follows:

TREASURER'S REPORT

Receipts

Cash on hand in bank January 1, 1914	$454.32	
Interest on bank deposits, January 1 to December 1	15.49	
5 vols. and 1 no. PHYTOPATHOLOGY	15.50	
266 members' dues, 1914	798.00	
Back dues, 1913	3.00	
Exchange on checks and sale of membership lists	1.24	
Cash contribution from Dr. Farlow for PHYTOPATHOLOGY	50.00	$1337.55

Disbursements

Printing, postage, expressage, etc	23.74	
300 separates of abstracts, Atlanta meeting	35.00	
Clerical assistance	20.25	
Williams and Wilkins, 5 vols. and 1 no. PHYTOPATHOLOGY	15.50	
To 367 members, subscriptions of PHYTOPATHOLOGY	734.00	828.49
Balance on hand		$509.06

The financial statement of the business manager of PHYTOPATHOLOGY, Donald Reddick, was presented and referred to an auditing committee consisting of H. R. Fulton and F. C. Stewart, which examined and approved the accounts. A small deficit was shown when the December bills which were unpaid were deducted.

RESOLUTIONS

The Society received greetings by telegraph from the newly organized Western Branch, meeting at Corvallis, Oregon. The following resolution was presented and adopted:

The Council recommends that the Society extend cordial greetings to the newly organized Western Branch of the American Phytopathological Society. It also recommends that a committee consisting of the present president, Haven Metcalf, and the secretary, with power to increase their number to five, be authorized to formulate the necessary terms of affiliation to provide for this and any other future branches which may be organized.

WHEREAS, it is known that a serious disease-producing parasite (*Urophlyctis alfalfae*) occurs in certain sections of Europe and this country and is liable, unless checked, to spread in the near future to other sections to the permanent injury of this highly valuable crop, and

WHEREAS, the plant pathologists or other officials of the individual states are unable properly to meet the situation, partly from lack of information, partly because it is essentially an international and interstate problem, be it therefore

Resolved, that we respectfully invite the attention of the Honorable Secretary of Agriculture and other officials of the U. S. Department of Agriculture to the above facts and urge the importance of immediate and earnest investigation under their leadership as to the present occurrence and seriousness of the disease, as to its means of distribution and as to what steps, if any, should be taken to check its further spread.

Resolved, the American phytopathologists greatly appreciate the importance of Schweinitz' collection of fungi now in the possession of the Philadelphia Academy

of Natural Science, it being the earliest large American collection. All botanists are interested in its preservation and especially those doing monographic work on fungi.

It is recommended that a committee of three be appointed by the Chair to confer with the Academy with reference to any steps that may be desirable to thoroughly insure the permanent preservation of the collection.

C. L. Shear, J. C. Arthur, and A. G. Johnson were appointed.

Resolved, that The American Phytopathological Society records its pleasure at the opportunity of becoming personally acquainted with Dr. Otto Appel and its satisfaction with the outcome of this first formal attempt to establish closer international phytopathological relations. We wish hereby to express our appreciation of the courtesy and scientific spirit in which Dr. Appel has coöperated in the consideration of our problems, and express our hope that this is but the first of such undertakings by the U. S. Department of Agriculture.

Resolved, that the Society express its deep appreciation of the efficient and faithful service of the retiring chairman of the Board of Editors, Prof. L. R. Jones, and that it also express its confidence in its new editor, Dr. Donald Reddick, and assure him of its active support.

MOTIONS

Upon motion, all the acts of the Council as reported to the Society were approved and adopted.

Moved, that the second paragraph under Editorial Notices on the inside cover of PHYTOPATHOLOGY be changed to read as follows:

"Manuscripts should be typewritten and carefully revised before submission. While no attempt will be made at exact uniformity in capitalization, punctuation, spelling, citations, footnotes, etc., each contributor is requested to give careful attention to clearness and preciseness in these matters and to conform as far as practicable to the best usage in this JOURNAL."

Moved, that a committee of three be appointed by the Chair to confer with Professor Farlow, and, in its discretion, with any other persons concerned, to express the sentiment of the Society that the continued delay in the publication of the Bibliographical Index of North American Fungi is a distinct loss to science and particularly disadvantageous to plant pathology, and to ascertain if anything can be done to remove existing obstacles and to facilitate its early publication.

Dr. C. E. Bessey was appointed chairman of the Committee with power to select his associates.

H. S. Reed called the attention of the Society to the desirability of institutional standardization with reference to plant pathology. Upon motion the president was directed to appoint a committee to consider this matter.

H. S. Reed, H. H. Whetzel and H. R. Fulton were appointed.

A contribution of $25 toward the Millardet Memorial at Bordeaux, recommended by the Council, was withdrawn, as it was learned that the memorial had already been completed and paid for.

It was voted to increase the price of back volumes of PHYTOPATHOLOGY to $4 after July 1, 1915. It is hoped that all the members and others wishing to fill out sets will take advantage of this opportunity and purchase before the price is increased.

On recommendation of the Council the Society voted to appropriate $200, or as much thereof as needed from the funds of the Society, toward the expenses of PHYTOPATHOLOGY, during the year 1915.

The following constitutional amendment proposed at the last meeting was adopted:

Article 3, section 3, shall be changed to read, "Any person may become a patron upon the payment of $100."

The Society passed a unanimous vote of thanks to the Local Committee for the excellent facilities and courtesies offered the Society during the meeting and to Dr. F. D. Heald for the care of the exhibits and other assistance in promoting the success of the meeting, also to the Chair for conducting the meetings with promptness and carrying the program through on time.

The method of presenting papers by abstract introduced at the Atlanta meeting was continued, with slight modifications, at the Philadelphia meeting, with great success. Six minutes were allowed for the presentation of each paper, the author being permitted to read the abstract as printed or use the allotted time in giving additional explanation or presentation of the topic, after which five minutes were allowed for discussion. The same method of handling the program was adopted for the future and the secretary authorized to limit the time for the acceptance of titles and abstracts, and to accept no titles for the program which are not included in the printed abstracts. Abstracts should be restricted to 200 words and must be in the hands of the secretary by December 1 in order that they may be published in the December issue of PHYTOPATHOLOGY.

C. L. SHEAR,
Secretary-Treasurer

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